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Letters

What causes dyslexia?: comment on Goswami

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Finding an underlying deficit that links the disparate impairments associated with dyslexia would be major breakthrough. In a recent article in *TiCS*, Goswami [1] offers a viable candidate for such a deficit – and has done a remarkable job of finding links that are plausible if still mostly circumstantial. Her stimulating article raises numerous directions for future research.

The epidemiology of dyslexia is poorly documented. Although many deficits have been reported [2], how often they occur and co-occur at different ages in different languages is still unknown. Goswami's descriptions of what is found in dyslexia should be read as what was found in some studies. More facts are needed; for example, with respect to the greater sensitivity of dyslexics to allophonic variation (which is crucial to Goswami's account of phoneme-level difficulties), many have sought this effect, and some have found it. The null results, however, get filed away.

Studies testing the Temporal Sampling Framework (TSF) need to be conducted more widely and with a variety of methods, together with tests of other putative deficits. The challenge is not only to establish closer mechanistic, causal connections between the hypothesized deficit and diverse behavioral impairments, but also to explain the distribution of impairments. The theory also needs to accommodate strong evidence for mainly left-hemisphere subcortical anomalies in dyslexia [3,4].

Short of a large-scale international epidemiological study, researchers (both of brain and behavior) need to test the same subjects using each other's measures, and post all results, both positive and negative. An online archive in which researchers could deposit their stimulus materials would make this possible. Perhaps a major funding agency could see the value in this undertaking.

Because reading is a complex task drawing on numerous capacities, it is unsurprising that multiple genetic polymorphisms are apparently involved. The TSF would be stronger if there were a reason why various genetic anomalies converge on low-frequency oscillation in the right hemisphere.

I would be inclined to search for anomalies in brain development that have, as one highly salient consequence, the deficit that Goswami has identified. I present the following speculative sketch to illustrate the type of multilevel theory to which we might aspire.

(i) Prominent candidate genes for dyslexia are implicated in cell migration [5].
(ii) Disorders of brain development often involve disturbances of interneuron migration and integration [6].
(iii) Anomalies in the migration of GABAergic (inhibitory) interneurons may underlie a variety of developmental disorders. Such anomalies can be regional rather than global [7].
(iv) GABAergic interneuron pathology impairs lateral inhibition, affecting discrimination of competing types of sensory information [8].
(v) Auditory processing at multiple time and frequency scales in parallel requires resolution of such competing information, and similarly for vision.
(vi) For some unknown reason, the processing of lower temporal frequency auditory information is particularly vulnerable,
(vii) From which deficits on tasks that rely on this information follow.

Therefore, rather than the auditory-processing deficit causing associated impairments in vision, motor performance, attention, learning, memory, and so on, all these impairments arise from a common source: an attested type of neurodevelopmental anomaly, caused by multiple genes, creating the observed variability in the phenotypic outcome. Processing of low temporal frequency auditory signals might be especially affected, with multiple downstream consequences.

Goswami's article is an interesting addition to the literature and her theory will stimulate much valuable research. However, much remains to be learned.

References

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